Corrigendum

Dr. W. B. McClure of the Department of Health, Ontario, writes us to say that there was an error in his manuscript, entitled "Pneumococcus Typing in the Public Health Laboratory". His article appeared in the *Journal* for April, page 365. The first four lines of the last paragraph on this page should read:

"There were 584 sputa where pneumococci were found, of which 87 (15 per cent) were Type I; 21 (3.5 per cent) were Type II; and 77 (13 per cent) were Type III."

Special Articles

INTRATHORACIC DYNAMICS IN RELATION TO ARTIFICIAL PNEUMOTHORAX

(A REVIEW)

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Collapse therapy has become such an important part of the treatment of pulmonary tuberculosis that an understanding of the movements and pressure changes occurring or induced in the thorax is a necessary part of the mental equipment of all who treat tuberculosis.

HOW NEGATIVE INTRATHORACIC PRESSURE ARISES[1]

In the new-born babe the lung fills the thoracic cavity, and there is no negative intrapleural pressure. Each expiration empties the lungs and leaves practically no reserve air. This is compensated by a more perfect ventilation of alveoli. In the eight-days old infant the intrapleural pressure was found by Hermann to be 5.4 mm. of water. The negative pressure of the adult gradually develops because the size of the thorax increases more rapidly than the size of the lungs as the child grows. The reserve air is created gradually at the expense of ventilation of the alveoli, which is less perfect in the adult than in the infant.

Observations of intrapleural pressure are almost lacking in early infancy, but reserve airspace must increase rather rapidly because pneumothorax therapy has been given successfully to a 10 months old infant[2] and no comment was made on the pressure readings obtained.

NORMAL INTRATHORACIC PRESSURES

Normally the negative intrapleural pressure is created by the tendency to recoil of the elastic tissue of the lung. Therefore measurements of the intrapleural pressures in the *normal* chest are measurements of the pull exerted in various phases of respiration by this pulmonary elastic tissue. The negative intrapleural pressure is the easiest intrathoracic pressure to measure, but it should be remembered that the intramediastinal pressure is also negative, its negativity no doubt reduced somewhat by the resistance of the mediastinal septa. The occasion can hardly arise, apart from a deliberate experiment, to determine the pressure in the completely healthy pleural cavity. However a few such experiments have been performed. Normal intrapleural pressures have been stated by various authors as in Table I.

TABLE I.
INTRAPLEURAL PRESSURES IN MM. WATER

Author	*Inspiration*	*Expiration*
Aron[3]	−63	−40.9
Macleod[4]	−135.5	−67.75
Hennell[1]	−70 to −90	−30 to −50

Dobbie[5] has called attention to the difference which exists between manometer readings and actual pressures in the pleural cavity. For example, if the difference in height between the fluid levels in the two limbs of an open manometer is 10 cm. then $X = A + \frac{10}{1033}$ of an atmosphere. (When X = pressure on the surface of the lower fluid level and A = pressure of one atmosphere.) An atmosphere is the equivalent of the weight of a column of water 76 × 13.6 cm. in height. Therefore each centimetre of height of the column of water in the manometer represents $\frac{1}{76 \times 13.6} = \frac{1}{1033}$ of an atmosphere; and 10 cm. $= \frac{10}{1033}$ of an atmosphere. In round numbers $X = A + \frac{1A}{100} = \frac{101A}{100}$. If the difference in levels be 20 cm. this formula becomes $X = \frac{102A}{100}$. Obviously, doubling the difference in levels does not double the pressure. Nor does doubling the reading given in one limb of the manometer give the true pressure.

One should remember too that manometer readings are markedly influenced by such factors as the bore of the needle, the bore of the manometer, and the permeability of the filter (if filter be between chest and manometer). These factors do not affect mean pressures, but do influence the extent of manometer oscillations.

Any alteration in the size of the hemithorax, by altering the degree of stretching of the elastic tissue of the lung, will alter the intrapleural pressure. The size of the hemithorax is normally altered in the following ways: (1) by the respiratory movements of the bony chest walls; (2) by alterations in the position of the diaphragm; (3) by shifting of the mediastinum to one side or the other.

In the expiratory phase the chest is smaller, the pulmonary elastic tissue is partially relaxed, and the intrapleural pressure is therefore less

strongly negative. In the inspiratory phase the intrapleural pressure becomes more strongly negative.

The position of the diaphragm of course varies with the phases of respiration, but in addition to this its position is affected by the degree of fullness of the abdomen, and by posture. When these causes operate to raise the diaphragm intrapleural pressure becomes less strongly negative. Woodcock,[6] at a post mortem, found an intrapleural reading of –1.5 inches of water. After puncturing the bowels to let gas escape, the intrapleural pressure was –3.5 inches.

In health, and especially in youth, the mediastinum is freely movable within limits which may be expected to vary considerably. A swing of the mediastinum to one side may be expected to reduce the negativity of the intrapleural pressures on that side provided other factors remain constant. Burrell and MacNalty,[7] however, quote two cases to show that this change in pressures does not always occur when air is injected into the opposite pleural cavity, even if the mediastinum be displaced.

Intrapleural pressure in health is not only affected by changes in the size of the hemithorax but also depends upon freedom of ingress and egress of air to and from the lungs. When a forced inspiration is made with the glottis closed a very large negative pressure will be produced in the pleural cavity, amounting according to MacLeod[4] to as much as –70 to –80 mm. of mercury. In the opposite condition in which the trachea is blocked and a forced expiration made, as for example during the first stage of coughing or during such acts as parturition or defæcation, or when an effort is made to blow against resistance, a strongly positive pressure in the pleural cavity may be observed. (Obviously, the use of "blow bottles" to expand a collapsed lung in closed pneumothorax is based on faulty theory. The water should be shifted from one bottle to the other by forced inspiration—not by forced expiration).

Pressures in the trachea are of interest, because on them the aeration of the collapsed lung largely depends. West (quoted by Emerson[11]) gives them as –0.5 to +3 mm. of mercury or –7 to +41 mm. of water on inspiration and expiration respectively.

EFFECT OF POSTURE ON INTRAPLEURAL PRESSURES

The intrathoracic volume in the dorsal recumbent position is less than in the erect position. This reduction in intrathoracic volume of course renders intrapleural pressure less negative. In the dorsal recumbent position the diaphragm rises somewhat. When lying on the pneumo side the pressures in the pneumo cavity are higher than when in the dorsal recumbent position or when lying with the pneumo uppermost. Burrell in one case noted a postural difference in pressure as great as the difference in pressure produced by the injection of 450 c.c. of air in that particular case. Lawson[8] observed postural pressure differences of "10 cm. of water or more". Fisher[9] notes that the ascent of the dependent diaphragm is accentuated if phrenicectomy has been performed on this side. Postural treatment may therefore be used to enhance the effect of this form, indeed of all forms of collapse therapy.

EFFECT OF ALTITUDE ON INTRAPLEURAL PRESSURES

Mantoux[10] sent a patient from an altitude of 1,050 metres to one of 1,914 and back. Thirty-five minutes before the patient set out on his trip his intrapleural pressure as recorded by the manometer was +9, and twenty minutes after arriving at the higher altitude after a journey of an hour and three-quarters the pressure was +17. He was back at the starting point within three hours of leaving it, and the pressure was found to be zero. The difference of 900 metres in altitude results in a difference of about 7.5 cm. of mercury in barometric pressure, or 102 cm. of water. The difference in intrapleural pressure is of course only a small fraction of this. But certainly some enlargement of the pneumo cavity will occur with increase in altitude. One would expect that changes in altitude would be more poorly borne by patients in whom pleural thickening is present, because the rate of diffusion of the gas will be slowed.

INTRAPLEURAL PRESSURES IN DISEASE

Pulmonary disease results in changes in lung volume and expansibility and contractibility in the diseased portion of the lung and in the healthy portions as well. When diseased portions of lung lose volume, as is usually the case, the healthy parts of the lung must stretch to make up for the shrinkage of diseased parts. The elastic tension of the healthy parts of the lung therefore is increased, and intrapleural pressures become more highly negative. However, it is possible for the healthy lung to be overstretched, in which case there will be a loss of elasticity and intrapleural negative pressure may in time become normal or subnormal. Thus even in diseased lungs the intrapleural pressure may be a measure of pulmonary elasticity. The presence of adhesions however in such cases often interferes with such a measurement.

In the type of compensatory emphysema described above there may be no loss of pulmonary elasticity. But in generalized bilateral and truly pathological emphysema pulmonary elasticity is reduced and there is a corresponding reduction in the negativity of intrapleural pressures. This permits the diaphragm to be pulled down by the weight of the subjacent organs, thus creating further overstretching of the alveolar walls by enlarging the thoracic cavity. There is no "pushing down of the diaphragm by voluminous lungs". There is therefore

theoretical justification for the use of an abdominal binder to keep the diaphragm elevated in forms of pulmonary disease resulting in decreased contractility of pulmonary tissue. The use of such a binder of course does not increase lung elasticity, but may help to prevent further reduction of elasticity, and should increase the excursion of the diaphragm and hence improve the ventilation of the lungs.

Since spontaneous or accidental pneumothorax is a fairly common complication of therapeutic pneumothorax it is important to understand the pressure changes it produces. The subject is well discussed in Emerson's notable monograph and recent years have added nothing to what is there so well set forth. Emerson[11] finds that a fresh wound in the lung is always valvular so long as it remains open. Therefore, following a lung perforation each inspiratory movement sucks air into the pleural cavity, and this air is prevented from escaping by the valvular action of the vent. Therefore intrapleural pressure increases towards the positive side and the mean pressure becomes positive. The pressure however can increase only till it equals the maximum pressure possible in the bronchial tree. If the vent is freely open the pressures will oscillate about zero; and after the vent closes, intrapleural pressure quite rapidly becomes negative in both phases of respiration. Accidental pneumothorax is therefore not ruled out by the finding of negative pressures.

PARIETAL PNEUMOTHORAX

Pneumothorax will develop following any perforation of the parietal pleura, no matter how small, for there is little cohesion of the pleural layers, provided of course that adhesions do not prevent retraction of the lung and that the opening in the parietal pleura remains open. Emerson gives the following description of the development of pneumothorax. "If the perforation be a parietal one and the diameter of the opening be less than that of the (main) bronchus to that lung, the air will enter with each inspiration, and during expiration some, but a less amount, will be expelled, until the lung at the end of expiration is just collapsed. Then with each succeeding breath the lung will expand somewhat with inspiration, since more air can enter through the bronchus to fill the partial vacuum than through the perforation. With each expiration the lung will resume its former collapsed condition since the air escapes more easily from the bronchus. The smaller the hole, the greater the expansion of the lung, and in case it be closed the lung can take no small part in respiration", provided of course the bronchi remain patent.

In "open pneumothorax", in which the external hole is larger than the main bronchus, there are no volume changes in the collapsed lung.[14] The swinging movements of the mediastinum with respiration will however continue.

THE DEVELOPMENT OF PNEUMOTHORAX

There is some difference of opinion as to the manner in which adhesion-free lung collapses during the induction of pneumothorax. Ehrenburg's[13] description seems to be the most acceptable. The lung may be considered as comprised of three zones. The "root zone" is almost incompressible, the "intermediate zone" contains both rigid and collapsible structures, and the "outer zone", 25 or 30 mm. in depth, is almost wholly elastic. Air injected into the pleural cavity becomes rarefied, and lighter than air in the alveoli, and so is buoyed up to the apex of the pleural cavity, or to the highest accessible point. Shifting of this air bubble may be felt by the patient if he bends over quickly, or perceived by the examiner's hand on the front of the chest. This phenomenon was noted first by Rist and Hirschfield and called by them "ballottement pulmonaire". The pneumothorax cavity, therefore, will have a conical shape with the base of the cone at the apex of the pleural cavity. Since tuberculosis commonly develops at the apex this localization of air will permit the lesion to drop down under the influence of gravity, giving the appearance of selective collapse. Once the diseased area is collapsed it cannot expand as readily as healthy lung, so that later localization of air is really determined in large part by the expansibility of the various parts of the lung. Ehrenburg's description is supported by Emerson's experimental work on this subject.

MECHANISM OF RE-EXPANSION OF THE LUNG

Very little has been written on this aspect of pneumothorax. In fact, the only mention I could find in the literature was an abstract of an article by Reineboth[12] in Emerson's notable monograph. What follows is from this abstract.

The forces concerned in the expansion of the lung are: (1) forced expiration might be used to expand the lung if there be a hole in the thoracic wall and air can be forced out of the pleural cavity and kept out by a valve at the hole; (2) forced inspiration will stretch the lung by increasing the negative pressure in the pleural cavity; (3) adhesions, in the form of adhesive pleuritis, may cause re-expansion of the lung; (4) absorption of air; (5) loss of lung elasticity.

Obstacles to expansion may be: (1) adhesion of the collapsed lung about the hilus; (2) thickened pleura; (3) atelectasis and pulmonary fibrosis; (4) bronchial obstruction.

THE RESPIRATORY MOVEMENTS OF A COLLAPSED LUNG

The completely collapsed lung is airless. This condition of the collapsed lung could not occur if the bronchi remain patent. So long

as bronchi remain patent and the pneumothorax is closed the lung will participate to some extent in the movements and function of respiration. But when bronchi become plugged with exudate the lung becomes airless and will no longer play any part in respiratory function, nor will it take part in any movements save those transmitted to it by the heart and mediastinum. This state of affairs can exist in the presence of negative pressure pneumothorax. The movement of the collapsed lung therefore depends on the degree of aeration of the lung and, to a minor degree, upon the intrapleural pressure.

It is worth a separate paragraph to emphasize that the means of producing a completely airless lung is bronchial obstruction—not external compression.

It is true however, that in the absence of adhesions, positive pressure pneumothorax will usually attain this result more rapidly than negative pressure pneumothorax.

The lung in a state of partial collapse, whether with or without adhesions, moves considerably during respiration, the healthy parts of the lung, however, move more than the diseased parts, because the power of the diseased parts to expand is lessened or abolished by disease. Obviously, too, there is a lessened contractility of diseased parts of the lung, so that these parts tend to resist collapse. Therefore, in the usual negative pressure pneumothorax most of the collapse is collapse of healthy lung. We are collapsing a zone of healthy tissues about diseased areas.

THE MOVEMENTS OF THE CHEST WALL DURING PNEUMOTHORAX

Little study of this has been made, and the few observations are not in agreement. Dufault[14] found that the chest wall moved almost as much on the pneumothorax side as on the other, no matter what the intrapleural pressure might be. He found diaphragmatic movement diminished and its anatomical position depressed if high pressures were used.

THE MOVEMENTS OF THE MEDIASTINUM IN PNEUMOTHORAX

One often hears it said that the presence of air or fluid in one pleural cavity results in the heart being "drawn over to the healthy side by the negative pressure in the pleural cavity". As Emerson points out, it is more correct to say that "The heart will move towards the sound side, pushed in that direction by a force equal to the difference in pressures in the two pleural cavities." There can be no drawing unless adhesions are present, and of course there is no movement at all of the mediastinum if it has been anchored by previous inflammation.

In pneumothorax and hydrothorax a pendulum movement of the mediastinum accompanies the rhythm of respiration. In inspiration air enters the uncollapsed lung more quickly than it enters the collapsed lung, and the mediastinum therefore swings in inspiration towards the pneumothorax side and swings back again in expiration.

THE PRESSURE CHANGES IN THE OPPOSITE PLEURAL CAVITY IN ARTIFICIAL PNEUMOTHORAX

The question of how much change in pressure in one pleural cavity affects pressure in the other is important, especially in cases of bilateral artificial pneumothorax. The answer depends upon whether or not mediastinal movement is possible. Bendove's[15] conception is that the thorax in man is divided into three vertical compartments by the mediastinal septa. When the pressure in one pleural cavity is raised, the pressure in the opposite cavity equals the intrapleural pressure in the pneumo cavity minus the elastic tension of both mediastinal septa. Simon,[16] using a goat, made some actual readings of pressures in both pleural cavities, injecting air on one side only. Table II shows an example of his results.

TABLE II.

Amount of air injected into pleural cavity (goat)	*Pleural cavity*	*Water manometer readings*
Before injection	RT	0.0, –1.5
	LT	0.0, –1.5
After giving 50 c.c.....	RT	4.0, –2
	LT	2.0, –2.5
10 minutes after giving second 50 c.c.	RT	4.5, –2
	LT	4.0, –3

He noted a similar result in 3 cases in men, and concluded that readings in the opposite pleural space in health follow the readings on the pneumothorax side quite closely. His findings are confirmed by Graham and Bell[17] and by Graham,[18] who notes that an alteration in pressure in one pleural cavity affects the intrapleural pressure on the opposite side almost equally up to a certain limit, where the difference in the two sides is governed by the degree of mediastinal displacement possible in the individual case.

MEDIASTINAL HERNIA

As one would expect, the herniation is largest in expiration and smallest at inspiration.

PARADOXICAL MOVEMENTS

In 1898 Kienboeck noted that in pyopneumothorax the level of the fluid may be observed to ascend with inspiration although the diaphragm descends on the opposite side. In 1929 Udaondo and Vadone[19] wrote that the "Kienboeck phenomenon" had come to mean not only paradoxical movement of fluid but also of the hemidiaphragm. They studied this matter in 200 cases of artificial pneumothorax and made the following observations: (1) section of the phrenic nerve in a

pneumothorax case altered the diaphragmatic contour, *i.e.*, the diaphragm was not paralyzed before phrenicotomy; (2) before insufflation if the intrapleural pressure at expiration was not positive, no "Kienboeck phenomenon" was constantly present; (3) one can observe appearance or disappearance of the Kienboeck phenomenon by adding or aspirating sufficient air.

Their explanation of the paradoxical movement is as follows. It is not exactly that the diaphragm rises at inspiration, but that it descends at expiration, when pleural pressures are highest and the diaphragm is relaxed. At inspiration the diaphragm rises to its usual position during inspiratory contraction.

This explanation however is not the whole story, though it appears to be the correct explanation of paradoxical movement in positive pressure pneumothorax. Bittorf[20] in 1910 and others since have observed paradoxical movement of the diaphragm in negative pressure pneumothorax. In these cases the collapsed lung fails to expand appreciably on inspiration, and the mediastinum is fixed. Inspiratory pressures are highly negative and the diaphragm is *aspirated* into the chest. A normal person can demonstrate this behind a fluoroscope by making a strong inspiratory effort with the glottis closed, when ascent of both halves of the diaphragm will be seen.

One further explanation of paradoxical movement of a fluid level in hydropneumothorax is offered by Rist[21] and receives general agreement. The fluid rises on inspiration, because the inspiratory shift of the mediastinum towards the pneumo side narrows the pleural cavity so that the fluid level must rise. Paradoxical movements may therefore be classed as follows: (1) those occurring in positive pressure pneumos, due primarily to depression of diaphragm in expiration; (2) those occurring in negative pressure pneumos due to aspiration of the diaphragm, and occurring in the normal person "inhaling" forcibly with nose and mouth closed; this is an inspiratory upward aspiration of the diaphragm; (3) those occurring with a paralyzed hemi-diaphragm; (4) those paradoxical shifts of fluid level due to pendulum movement of the mediastinum.

PARADOXICAL PRESSURES IN ARTIFICIAL PNEUMOTHORAX

Paradoxical movement is not to be confused with paradoxical pressures. For example, after phrenicectomy paradoxical movement is present, paradoxical pressures are not. Stivelman[22] found 4 out of 600 pneumo cases with paradoxical pressures. All of these cases had a limited (partial) pneumothorax with an adhesion to or near the diaphragm below and others to the upper chest above. The lung edge is convex towards the mediastinum. When the ribs rise and the diaphragm sinks in inspiration, this convexity becomes straight, lessening the size of the pleural cavity, and therefore increasing the intrapleural pressure on inspiration. Parfitt and Crombie[23] noted paradoxical movement of the manometer in 5 of 63 cases.

THE PARFITT-CROMBIE PHENOMENON

They also noted the phenomenon of rapid rise in pressure when the breath was held at the end of inspiration in 13 of 63 cases. This is thought to be due to contraction of the abdominal muscles which often accompanies this manœuvre.

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